The number of patients with epilepsy who are eligible for surgical treatment has increased in the past decade. Amygdalohippocampectomy with or without ATL is considered the standard surgical treatment for medically refractory epilepsy originating from the mesial temporal lobe structures.26 The success rate as defined by a significant reduction in seizures occurs in up to 80% of patients with low perioperative morbidity.3,4,7

Diplopia is a potential neuroophthalmological complication of ATL. Authors of many reports have cited an approximately 15% incidence of postoperative diplopia, which is generally attributed to oculomotor nerve dysfunction.19,23,27 The present study was designed to prospectively examine patients undergoing ATL for the incidence and pattern of postoperative ophthalmoplegia. Visual field defects are also a common and well-known side effect of ATL, usually superior quadrantanopias and rarely disabling.

Clinical Material and Methods

Patient Population

Forty-seven patients diagnosed with medically refractory epilepsy of temporal lobe origin were randomly entered into this prospective study between 1992 and 1994. Participants ranged in age from 18 to 60 years. Any history of strabismus, amblyopia, prior eye muscle surgery, thyroid ophthalmopathy, or myasthenia gravis excluded participation. A remote history of diplopia did not exclude patients from the study given that diplopia is a relatively common side effect of all anticonvulsant drugs administered at therapeutic levels.20 Patients with epilepsy related to a neoplasm demonstrated on MR imaging or on pathological evaluation were also excluded. Mesial temporal lobe sclerosis was the most common preoperative diagnosis based on MR imaging studies.25 Postoperative MR images were obtained using the same magnet with and without infusion of Gd contrast. Each patient gave informed consent to participate in the research protocol, which had been approved by the Mayo Clinic Institutional Review Board.

Patients were reexamined within the 1st postoperative week if possible. Those with any complaints of diplopia underwent further Lancaster red green testing as well as measurements with head tilts to the right and left. This group was reexamined 3 to 6 months postoperatively. Serum levels of anticonvulsant drugs were recorded at the time of preoperative and postoperative eye examinations.

Ophthalmological Examination

All study patients underwent a preoperative ophthalmological examination, including vision, intraocular pressure measurement, stereocuity measurement, palpebral fissure...
measurement, pupillary measurement (in dim and bright light), vertical fusional amplitudes, alternate cover, Lancaster red green, and computer automated visual field testing. Any patient with evidence of a preexisting strabismus or ophthalmic cranial nerve palsy on preoperative examination was excluded.

Stereocuity measurements of depth perception were obtained using a set of polarized glasses to view images with decreasing degrees of crossed disparity. The images with crossed disparity would appear to be in front of the reference plane. Normal stereoscopic discrimination (40 seconds of arc) was possible only with normal binocular fusion, thereby indicating those with strabismus or amblyopia.

Palpebral fissure measurement and pupillary examinations were performed to evaluate the levator palpebrae and pupillary sphincter muscles innervated by the oculomotor nerve.

Vertical fusional amplitudes were performed to distinguish congenital trochlear nerve palsy. A vertical prism was placed over one eye until diplopia was induced. The strength of the prism was reduced until the images became one. The prism power required to create diplopia would be greater than 6 to 10 prism diopters in people with congenital trochlear nerve palsy. Many patients with congenital trochlear nerve palsy do not become symptomatic until the third or fourth decade of life or following minor head trauma.

Alternate cover testing will break binocular fusion to reveal ocular deviations. The Lancaster red green testing is a standardized test with reproducible results, which is used to determine the pattern and amount of ocular deviation in seven positions of gaze. 2 The patient performs the test while looking through a pair of glasses with one red and one green lens. These glasses prevent binocular fusion. Without binocular fusion the deviation measured is a summation of both manifest tropias and latent phorias. The test is conducted twice, once with each eye fixating. The pattern of ophthalmoplegia on ocular examination was excluded.

Any patient with evidence of a preexisting strabismus or ophthalmic cranial nerve palsy on preoperative examination was excluded.

Six patients revealed that the postoperative diplopia following surgery was caused by trochlear nerve palsy. The palsy always occurred on the side ipsilateral to the site of surgery. In all cases, the diplopia and trochlear nerve palsy had completely resolved within 3 to 6 months after surgery.

The pattern of ophthalmoplegia on ocular examination and Lancaster red green testing demonstrated no evidence of oculomotor nerve dysfunction. None of the patients had limitation of their medial, inferior, or superior rectus muscles. Mild ptosis was not uncommon because of edema of the upper lid immediately after surgery. All anticonvulsant drug levels were within normal therapeutic range at follow-up examinations. In the patients with diplopia, postoperative MR images revealed no notable findings other than mesial temporal sclerosis in every case, showing gliosis and neuronal loss.

Discussion

Temporal lobectomy and amygdalohippocampectomy are associated with an overall low morbidity. 26 The most common postoperative complication of ATL is a contralateral superior quadrantanopia. 19 Memory difficulties, vascular injury, dysphasia, hemiparesis, and diplopia are less common. In a retrospective review by Jacobson, et al., 9 three (14%) of 22 patients who underwent ATL experienced diplopia caused by trochlear nerve palsy immediately after surgery. Data from our prospective study of 47 patients revealed that the postoperative diplopia following
ATL is caused by trochlear nerve involvement and occurs in up to 19% of cases.

Traction, compression, bipolar electrocautery, contact with dissecting instruments, or exposure to blood and other fluids at undesirable temperatures can account for postoperative cranial nerve deficits. These factors presumably cause segmental demyelination of the nerve, compromised microcirculation, and postoperative edema. If demyelinated, the nerve recovers by remyelination within days or weeks. Ischemic nerve injury can also play a role. Cranial nerves do not contain epineurium to resist traction. Hence, any stretch on the nerve may compromise intraneural microcirculation. Trochlear nerve palsy with a presumed vascular cause is reportedly transient and seems to resolve within a 4-month period. Any of these mechanisms may account for the transient fourth cranial nerve palsy following ATL. Transient fourth nerve palsy has also been reported following lumbar puncture, myelography, spinal anesthesia, and placement of the foramen ovale electrodes for epilepsy evaluation. Authors attributed this side effect to the sudden decrease in cerebrospinal fluid pressure after the puncture and subsequent mechanical irritation of the nerve from the abrupt contact with the tentorium. This mechanism may also play a role in our series, because significant cerebrospinal fluid egress occurs after opening the temporal horn of the lateral ventricle during ATL.

Multiple other factors may account for the occurrence of transient trochlear nerve palsy following ATL. The trochlear nerve may be injured during resection of the mesial temporal structures due to the breach of the arachnoid membrane barrier overlying the ambient cistern. Compared with the oculomotor nerve, the trochlear nerve is less visible and therefore may be more vulnerable to injury given its long path along the free border of tentorium. Despite respecting the overlying mesial arachnoid membranes, extensive hippocampectomy posterior to the level of the tectal plate exposes this nerve to more harm. There is evidence to support aggressive total hippocampectomy (to the level of the colliculi) to provide increased seizure control, with no additional risk to memory. Observance of meticulous microsurgical techniques to prevent traction on the arachnoid membrane overlying the free border of tentorium may reduce the chance of trochlear nerve injury.

Trochlear nerve palsy after ATL in our series may be attributed to another factor. A self-retaining retractor was gently placed along the posterior and superior edges of cortical resection to enhance removal of the hippocampus tail. Patients have tolerated this maneuver well and we have not experienced any related complications such as retraction injury. The slight posterior displacement of the temporal lobe may indirectly distort the trochlear nerve by placing traction on the medial arachnoid membrane encasing this nerve. We have been able to reproduce this phenomenon in unfixed brain specimens extracted from cadavers. Traction nerve injury may also occur as the trochlear nerve is coursing in the lateral wall of the CS. Distortion of the wall during removal of the anteromedial temporal cortex may deform this nerve. Similarly, fourth nerve palsy during pregnancy has been attributed to the expansion and distortion of the wall of the CS due to the increased intravascular volume. Overall, indirect traction injury is the most plausible factor explaining this complication.

Fourth nerve palsy following ATL can be underreported in retrospective studies without controlled pre- and postoperative examinations. Diplopia is a relatively common side effect of anticonvulsant medications administered at therapeutic levels. Patients undergoing ATL often receive additional doses of their antiepileptic medication to reduce postoperative seizures. Without an appropriate ophthalmological examination to diagnose a superior oblique palsy, postoperative diplopia may be erroneously attributed to the higher levels of antiepileptic medications. Based on our prospective experience, we propose that a fourth nerve palsy after ATL occurs more frequently than thought previously. Fortunately, this palsy is transient and all patients recover within 3 to 6 months.

Conclusions

The primary goal of this study was to estimate the incidence and pattern of diplopia in the patients who underwent ATL. All patients with postoperative diplopia were found to have trochlear nerve palsy on ophthalmological examination. This may be corrected with an eye patch or temporary prismatic glasses. Patients also may compensate by adapting a head tilt to the contralateral side. Careful microsurgical techniques during resection of mesial temporal lobe structures may minimize this complication. It is important to realize that this deficit is transient; all patients we studied had resolution of their symptoms within 6 months after surgery.

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References

Diplopia after temporal lobectomy


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